QUIZ NAVIGATION

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Started on	Tuesday, 15 October 2024, 12:39 AM
State	Finished
Completed on	Tuesday, 15 October 2024, 12:58 AM
Time taken	19 mins 2 secs
Grade	**10.00** out of 10.00 (**100**%)

Question 1

ID: 39200

Correct

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THE NEXT 3 QUESTIONS INCLUSIVE REFER TO THE FOLLOWING CASE:

JK is a 56-year-old male (weighs 88 kg) who presented to the hospital with complaints of severe upper right abdominal pain and fever. The attending physician diagnosed JK with a biliary tract infection based on his symptoms. The following laboratory data was obtained:

- **Temperature: 39.5 °C**
- **WBC Count: 14 x 10^9 cells/L**
- **Respiratory rate: 18 breaths per minute**
- **Heart rate: 95 bpm**

A gallbladder ultrasound indicated gallstone formation. JK was admitted to the hospital, the stones were removed, and ciprofloxacin was started. Approximately 24 hours later, JK is still feverish and in pain. JK is also vomiting, his serum creatinine (SCr) has increased to 220 umol/L from baseline SCr of 100 umol/L, and he has a urine output of 100 mL/day.

JK's past medical history includes hypertension, hyperlipidemia, and osteoarthritis. He takes ramipril 5 mg daily, hydrochlorothiazide 25 mg daily, simvastatin 40 mg daily and ibuprofen 400 mg TID.

All of the following options describe the clinical presentation of an acute kidney injury (AKI), **EXCEPT**:

Select one:

- Vomiting ✖
- Reduced urine output ✖
- Upper quadrant abdominal pain ✔ — *Rose Wang (ID:113212) this answer is correct. Upper quadrant abdominal pain is not a sign of acute kidney injury (AKI).*
- Rise in serum creatinine ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury

LEARNING OBJECTIVE:

To identify the signs and symptoms of acute kidney injury (AKI).

BACKGROUND:

Acute kidney injury (AKI) occurs when there is a rapid decrease in kidney function. AKI can be classified based on where the damage or impairment is occurring in the kidney:

- *Pre-renal AKI* occurs when there is hypoperfusion of the kidney (the most common cause of AKI)
- *Intrinsic AKI* occurs when there is damage or impairment within the kidney
- *Post-renal AKI* occurs when a severe blockage beyond the kidney (e.g., in the ureter) causes waste buildup in the kidney

Possible causes of AKI

Impairment Location	Possible causes of AKI
Pre-renal AKI	• Hypovolemia • Increased vascular resistance • Reduced cardiac function • Systemic vasodilation
Intrinsic AKI	• Bilateral renal artery stenosis • Infection • Immune system dysfunction (e.g., lupus, IgA glomerulonephritis) • Nephrotoxic drugs

Post-renal AKI	▪ Blood clots ▪ Improperly placed catheter ▪ Kidney stones (nephrolithiasis) ▪ Urogenital cancers

AKI is defined as any of the following:

- Increase in SCr by ≥26.5 umol/L within 48 hours, or
- Increase in SCr by ≥1.5 times the baseline within 7 days, or
- Urine volume of <0.5 ml/kg/hour for 6 hours

In addition to reduced or lack of urine output, fluid retention is a very common sign of AKI. Nausea/vomiting can also occur as AKI results in a buildup of waste in the blood.

RATIONALE:

Correct Answer:

(Option #3): Upper quadrant abdominal pain is not a sign of acute kidney injury (AKI).

Incorrect Answers:

(Option #1): This is a common clinical presentation of acute kidney injury (AKI).

(Option #2): This is a common clinical presentation of acute kidney injury (AKI).

(Option #4): This is a common clinical presentation of acute kidney injury (AKI).

TAKEAWAY/KEY POINTS:

A significant increase in serum creatinine (SCr) that occurs within hours to a few days, along with reduced urine output, are strong indicators of acute kidney injury (AKI).

REFERENCES:

[1] KDIGO clinical practice guidelines for acute kidney injury. *Kidney International Supplements*. 2012;2(1). doi:10.1038/kisup.2012.1.

[2] Makris K, Spanou L. Acute Kidney Injury: Definition, Pathophysiology and Clinical Phenotypes. *Clin Biochem Rev*. 2016;37(2):85-98.

The correct answer is: Upper quadrant abdominal pain

Question 2

ID: 38923

Correct

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JK's attending physician approaches you and asks which of his medications should be discontinued to reduce the risk of worsening the damage to the kidneys.

Which of JK's medications is the **LEAST** likely to cause acute kidney injury (AKI)?

Select one:

- Ramipril ✖
- Ciprofloxacin ✖
- Ibuprofen ✖
- Simvastatin ✔

Rose Wang (ID:113212) this answer is correct. Statins can cause kidney damage only if rhabdomyolysis occurs - a very rare side effect of statins.

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury

LEARNING OBJECTIVE:

To understand how common medications can negatively impact the kidneys.

BACKGROUND:

Acute kidney injury (AKI) occurs when there is a rapid decrease in kidney function. AKI can be classified based on where the damage or impairment is occurring in the kidney:

- *Pre-renal AKI* occurs when there is hypoperfusion of the kidney (the most common cause of AKI)
- *Intrinsic AKI* occurs when there is damage or impairment within the kidney
- *Post-renal AKI* occurs when a severe blockage beyond the kidney (e.g., in the ureter) causes waste buildup in the kidney

Possible causes of AKI

Impairment Location	Possible causes of AKI

Pre-renal AKI	• Hypovolemia • Increased vascular resistance • Reduced cardiac function • Systemic vasodilation
Intrinsic AKI	• Bilateral renal artery stenosis • Infection • Immune system dysfunction (e.g., lupus, IgA glomerulonephritis) • Nephrotoxic drugs
Post-renal AKI	• Blood clots • Improperly placed catheter • Kidney stones (nephrolithiasis) • Urogenital cancers

Medications can cause AKI via several mechanisms:

- Changing the blood flow rate within the glomerulus
  - e.g., nonsteroidal anti-inflammatory drugs (NSAIDs), angiotensin-converting enzyme inhibitors (ACEi)
- Inflammation within the glomerulus
  - e.g., NSAIDs, propylthiouracil
- Acute interstitial nephritis due to drug binding to antigens in the kidney
  - e.g., fluoroquinolones, diuretics
- Drug crystalizes and causes a urinary outflow blockage
  - e.g., ciprofloxacin, methotrexate
- Rhabdomyolysis
  - e.g., statins, benzodiazepines
- Inducing clots within small vessels of the kidney
  - e.g., clopidogrel, cyclosporine

RATIONALE:

Correct Answer:

(Option #4): Statins can cause kidney damage only if rhabdomyolysis occurs - a very rare side effect of statins.

Incorrect Answers:

(Option #1): ACE inhibitors decrease blood pressure and therefore cause hypoperfusion of the kidneys.
(Option #2): Fluoroquinolones are nephrotoxic and can cause intrinsic damage to the kidneys.
(Option #3): NSAIDs can reduce blood flow into the glomeruli by inhibiting prostaglandins that dilate the renal artery.

TAKEAWAY/KEY POINTS:

Several medications are known to cause acute kidney injury, such as ACE inhibitors, fluoroquinolones, and NSAIDs.

REFERENCES:

[1] KDIGO clinical practice guidelines for acute kidney injury. *Kidney International Supplements*. 2012;2(1). doi:10.1038/kisup.2012.1.

[2] Makris K, Spanou L. Acute Kidney Injury: Definition, Pathophysiology and Clinical Phenotypes. *Clin Biochem Rev*. 2016;37(2):85-98.

[3] Naughton C. Drug-induced nephrotoxicity. *Am Fam Physician*. 2008;78(6):743-750.

The correct answer is: Simvastatin

Question 3
ID: 38879
Correct
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The physician asks for your advice on a treatment plan for JK's acute kidney injury (AKI).

In addition to removing the nephrotoxic agents, which of the following options is an appropriate course of action?

Select one:

- Fluid resuscitation ✔ *Rose Wang (ID:113212) this answer is correct. Fluid resuscitation treats*

with IV normal saline — *hypoperfusion and ensures sufficient blood flow to the organs.*

- Initiate dopamine infusion ✖
- Insert a Foley catheter to promote urine flow ✖
- Dialysis to remove excess fluid ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To understand the treatment options for acute kidney injury (AKI).

BACKGROUND:

Acute Kidney Injury Staging

Stage	Serum Creatinine (SCr)	Urine output
1	1.5 - 1.9 times increase from baseline OR ≥26.5 umol/L increase from baseline	<0.5 ml/kg/hour for 6 - 12 hours
2	2.0 - 2.9 times increase from baseline	<0.5 ml/kg/hour for ≥12 hours
3	3.0 times increase from baseline OR Increase in SCr to ≥353.6 umol/L OR Renal replacement therapy required	<0.3 ml/kg/hour for ≥24 hours OR Anuria (i.e., no urine) for ≥12 hours

AKI staging helps with identifying the AKI severity and how aggressive to be with the treatment.

If AKI is a result of a nephrotoxic drug and the drug can be discontinued, it should be stopped right away. If stopping the drug is too risky (e.g., it is treating a severe infection and there isn't an alternative antibiotic available), then consider reducing the drug dose if possible.

In addition to stopping the cause of AKI, fluid resuscitation is also commonly used to treat AKI as it ensures proper blood perfusion throughout the body. The goal of fluid resuscitation is to achieve a mean arterial pressure of 65 - 90 mmHg.

Vasopressors may be given along with fluid resuscitation to treat vasomotor shock (i.e., systemic dilation of blood vessels).

Renal replacement therapy (RRT) is reserved only for individuals with life-threatening changes in fluid, electrolytes, and blood pH. RRT is stopped as soon as the individual's kidney function returns to a point where it meets the individual's needs, or goals of care have changed and RRT is no longer required.

RATIONALE:

Correct Answer:

(Option #1): Fluid resuscitation treats hypoperfusion and ensures sufficient blood flow to the organs.

Incorrect Answers:

(Option #2): Dopamine use is not recommended due to increased risk of arrhythmia.
(Option #3): Foley catheters may be used to treat post-renal obstruction only.
(Option #4): JK's creatinine clearance is still 40 mL/min and there are no other indications for dialysis.

TAKEAWAY/KEY POINTS:

In acute kidney injury (AKI), stop the nephrotoxic agent as soon as possible. In addition, systemic hypoperfusion is a likely complication of AKI and is treated with fluid resuscitation.

REFERENCE:

[1] KDIGO clinical practice guidelines for acute kidney injury. Kidney International Supplements. 2012;2(1). doi:10.1038/kisup.2012.1.

The correct answer is: Fluid resuscitation with IV normal saline

Question 4

ID: 38880

Correct

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THE NEXT 4 QUESTIONS INCLUSIVE REFER TO THE FOLLOWING CASE:

FT is a 73-year-old woman with heart failure (HF), hypertension (HTN), osteoporosis, diabetes, and hypothyroidism. She takes candesartan 16 mg daily, atorvastatin 40 mg daily, acetylsalicylic acid 81 mg daily, alendronate 10 mg daily, spironolactone 12.5 mg daily, metformin 1000 mg BID, bisoprolol 2.5 mg daily and levothyroxine 100 mcg daily.

FT presents to the hospital with severe weakness and reduced urine output. Blood samples reveal a potassium level of 6.1 mmol/L and a serum creatinine (SCr) of 240 umol/L. Her electrocardiogram (ECG) is normal.

Which of the following medications increases FT's risk of hyperkalemia?

Select one:

- Alendronate ✖
- Spironolactone ✔

Rose Wang (ID:113212) this answer is correct. Spironolactone limits potassium excretion and sodium reabsorption in nephrons by antagonizing aldosterone.

- Atorvastatin ✖
- Acetylsalicylic acid ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify the signs and causes of hyperkalemia as it can be a complication of renal dysfunction.

BACKGROUND:

Potassium is an electrolyte that is critical for proper functioning of smooth muscles, skeletal muscles, the heart, and nerves.

Normal potassium level is between 3.3 - 5.1 mmol/L. There isn't evidence to suggest the exact potassium level at which intervention is required. However, it is generally accepted that potassium ≥5.5 mmol/L requires non-pharmacological treatment and potassium ≥6.0 mmol/L requires pharmacological treatment.

Symptoms of hyperkalemia include:

- Muscle weakness
- Flaccid paralysis
- Partial or complete blockage of the intestine (ileus)
- Changes in ECG
  - Tall peaked T wave
  - Loss of P wave with tall peaked T wave
  - Widened QRS with tall T wave

Causes of Hyperkalemia

Lab work issue	Laboratory error
	Repeated clenching of the fist while blood is drawn
	Lysis of blood sample
Medication	Angiotensin-converting enzyme inhibitors (ACEi)
	Angiotensin II receptor blockers
	Azole antifungals
	Beta-blockers
	Cyclosporine
	Digoxin (at toxic levels)
	Glucose infusion
	Insulin deficiency
	Nonsteroidal anti-inflammatory drugs
	Potassium-sparing diuretics (amiloride, eplerenone, spironolactone, triamterene)
	Potassium supplements
	Tacrolimus
	Trimethoprim
Medical conditions	Chronic kidney dysfunction
	Renal failure
	Renal hypoperfusion
	Hypoaldosteronism

RATIONALE:

Correct Answer:

(Option #2): Spironolactone limits potassium excretion and sodium reabsorption in nephrons by antagonizing aldosterone.

Incorrect Answers:

(Option #1): Bisphosphonates are not associated with increased potassium levels.
(Option #3): Statins are not associated with increased potassium levels.
(Option #4): Low dose acetylsalicylic acid is not associated with increased potassium levels.

TAKEAWAY/KEY POINTS:

There is a long list of medications that can induce hyperkalemia and bloodwork should be done at regular

intervals to monitor potassium levels.

REFERENCES:

[1] Hollander-Rodriguez JC, Calvert JF. Hyperkalemia. *Am Fam Physician*. 2006;73(2):283-290.

[2] Elliot MJ, Ronksley PE, Clase CM, Ahmed SJ, Hemmelgarn BR. *Can Med Assoc J*. 2010;182(15):1631-1635. doi:https://doi.org/10.1503/cmaj.100461.

The correct answer is: Spironolactone

Question 5

ID: 38881

Correct

Flag question

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Which of the following options would be the most likely cause of FT's acute kidney injury (AKI)?

Select one:

- Heart failure ✔ *Rose Wang (ID: 113212) this answer is correct. Heart failure can cause hypoperfusion of the kidneys, which may lead to acute kidney injury (AKI).*
- Hypothyroidism ✖
- Osteoporosis ✖
- Hypercholesterolemia ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify the signs and causes of acute kidney injury (AKI).

BACKGROUND:

Acute kidney injury (AKI) occurs when there is a rapid decrease in kidney function. AKI can be classified based on where the damage or impairment is occurring in the kidney:

- *Pre-renal AKI* occurs when there is hypoperfusion of the kidney (the most common cause of AKI)
- *Intrinsic AKI* occurs when there is damage or impairment within the kidney
- *Post-renal AKI* occurs when a severe blockage beyond the kidney (e.g., in the ureter) causes waste buildup in the kidney

Possible causes of AKI

Impairment Location	Possible causes of AKI
Pre-renal AKI	• Hypovolemia • Increased vascular resistance • Reduced cardiac function (e.g., heart failure) • Systemic vasodilation
Intrinsic AKI	• Bilateral renal artery stenosis • Infection • Immune system dysfunction (e.g., lupus, IgA glomerulonephritis) • Nephrotoxic drugs
Post-renal AKI	• Blood clots • Improperly placed catheter • Kidney stones (nephrolithiasis) • Urogenital cancers

AKI is defined as any of the following:

- Increase in SCr by ≥26.5 umol/L within 48 hours, or
- Increase in SCr by ≥1.5 times the baseline within 7 days, or
- Urine volume of <0.5 ml/kg/hour for 6 hours

In addition to reduced or lack of urine output, fluid retention is a very common sign of AKI. Nausea/vomiting can also occur as AKI results in a buildup of waste in the blood.

RATIONALE:

Correct Answer:

(Option #1): Heart failure can cause hypoperfusion of the kidneys, which may lead to acute kidney injury (AKI).

Incorrect Answers:

(Option #2): Hypothyroidism is not a cause of acute kidney injury (AKI).
(Option #3): Osteoporosis is not a cause of acute kidney injury (AKI).
(Option #4): Hypercholesterolemia is not the most likely cause of the acute kidney injury (AKI).

TAKEAWAY/KEY POINTS:

A significant increase in serum creatinine (SCr) that occurs within hours to a few days, along with reduced urine output, are strong indicators of acute kidney injury (AKI). Heart failure can cause hypoperfusion of the kidneys, which may lead to AKI.

REFERENCES:

[1] KDIGO clinical practice guidelines for acute kidney injury. *Kidney International Supplements.* 2012;2(1). doi:10.1038/kisup.2012.1.

[2] Makris K, Spanou L. Acute Kidney Injury: Definition, Pathophysiology and Clinical Phenotypes. *Clin Biochem Rev.* 2016;37(2):85-98.

The correct answer is: Heart failure

Question 6

ID: 38882

Correct

Flag question

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Based on FT's clinical presentation, the doctor decides insulin plus dextrose is the most appropriate therapy.

What is the mechanism by which insulin treats hyperkalemia?

Select one:

- Insulin moves potassium extracellularly ✖
- Insulin moves potassium intracellularly ✔ *Rose Wang (ID: 113212) this answer is correct. Blood potassium level is reduced by increasing cellular uptake of the electrolyte.*
- Insulin increases renal potassium excretion ✖
- Insulin binds potassium as a cation for excretion ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify treatment options for hyperkalemia as it can present along with acute kidney injury (AKI).

BACKGROUND:

Hyperkalemia can be identified:

Hyperkalemia severity and treatment

Severity	Serum potassium level (K^+)	Treatment
Mild	5.5 - 5.9 mmol/L	Non-pharmacological treatment such as: ▪ Reduce dietary sources of K^+ ▪ Optimize control of disease condition causing hyperkalemia ▪ Temporary or permanent discontinuation of K^+ increasing drugs
Moderate	6.0 - 6.4 mmol/L	• Non-pharmacological treatment • Repeat serum K^+ and kidney function lab work within 3 days • If second serum K^+ result is within the same range, consider administration of cation binding agent
Severe	≥6.5 mmol/L	Treat pharmacologically as soon as possible if the patient is symptomatic or has abnormal electrocardiogram (ECG) results. Pharmacological treatments include: ▪ Insulin with glucose ▪ Inhaled, nebulized, or IV β_2 agonists ▪ Calcium carbonate ▪ Furosemide ▪ Cation binding agent

In addition to pharmacological treatment, use non-pharmacological treatment and repeat serum K^+ level within 3 days.

Hyperkalemia can be treated pharmacologically via different mechanisms:

Insulin

- Insulin stimulates the activity of sodium-hydrogen antiporter, resulting in intracellular movement of sodium; this activates the sodium-potassium ATPase pump, causing an intracellular shift of potassium

β2 agonists

- β2 agonists activate the sodium-potassium ATPase pump, causing an intracellular shift of potassium

Calcium carbonate

- Calcium does not reduce serum K^+ but reduces the risk of arrhythmia ECG changes present as a result of hyperkalemia

Furosemide

- Furosemide increases urinary excretion of K^+

Cation binding agent

- Cation binding agents such as sodium polystyrene sulfonate bind K^+ and release sodium into the gut, resulting in fecal excretion of bound K^+

RATIONALE:

Correct Answer:

(Option #2): Blood potassium level is reduced by increasing cellular uptake of the electrolyte.

Incorrect Answers:

(Option #1): Insulin does not move potassium extracellularly.
(Option #3): Insulin does not increase kidney excretion of potassium.
(Option #4): This is the action of cation exchange medications, not insulin.

TAKEAWAY/KEY POINTS:

There are multiple mechanisms for treating hyperkalemia.

REFERENCES:

[1] Ezekowitz JA, et al. 2017 Comprehensive update of the Canadian cardiovascular society guidelines for the management of heart failure. *Can J Cardiol*. 2017;33(11):1342-1433.

[2] Elliot MJ, Ronksley PE, Clase CM, Ahmed SB, Hemmelgarn BR. Management of patients with acute hyperkalemia. *Can Med Assoc J*. 2010;182(15):1631-1635. doi:https://doi.org/10.1503/cmaj.100461.

The correct answer is: Insulin moves potassium intracellularly

Question 7

ID: 38883

Correct

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FT's renal injury and hyperkalemia have been resolved and the doctor discharges her back to her long-term care home.

Which counselling point is most appropriate to reduce the risk of future hyperkalemia events for FT?

Select one:

- Recommend use of a daily multivitamin to balance electrolytes ✖
- Recommend regular bloodwork and electrocardiogram (ECG) ✖
- FT should discontinue the use of candesartan and spironolactone ✖
- Acetaminophen is the only safe over-the-counter oral analgesic for FT ✔

Rose Wang (ID:113212) this answer is correct. Acetaminophen is the only over-the-counter analgesic which will not increase potassium nor increase the risk of acute renal injury.

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify the signs and causes of hyperkalemia as it can be a complication of renal dysfunction.

BACKGROUND:

Potassium is an electrolyte that is critical for proper functioning of smooth muscles, skeletal muscles, the heart, and nerves.

Normal potassium level is between 3.3 - 5.1 mmol/L. There isn't evidence to suggest the exact potassium level at which intervention is required. However, it is generally accepted that potassium ≥5.5 mmol/L requires non-pharmacological treatment and potassium ≥6.0 mmol/L requires pharmacological treatment.

Symptoms of hyperkalemia include:

- Muscle weakness

- Flaccid paralysis
- Partial or complete blockage of the intestine (ileus)
- Changes in ECG
  - Tall peaked T wave
  - Loss of P wave with tall peaked T wave
  - Widened QRS with tall T wave

Causes of Hyperkalemia

Lab work issue	Laboratory error
	Repeated clenching of the fist while blood is drawn
	Lysis of blood sample
Medication	Angiotensin-converting enzyme inhibitors (ACEI)
	Angiotensin II receptor blockers
	Azole antifungals
	Beta-blockers
	Cyclosporine
	Digoxin (at toxic levels)
	Glucose infusion
	Insulin deficiency
	Nonsteroidal anti-inflammatory drugs (NSAIDs)
	Potassium-sparing diuretics (amiloride, eplerenone, spironolactone, triamterene)
	Potassium supplements
	Tacrolimus
	Trimethoprim
Medical conditions	Chronic kidney dysfunction
	Renal failure
	Renal hypoperfusion
	Hypoaldosteronism

RATIONALE:

Correct Answer:

(Option #4): Acetaminophen is the only over-the-counter analgesic which will not increase potassium nor increase the risk of acute renal injury.

Incorrect Answers:

(Option #1): Multivitamins often contain potassium and can increase serum potassium level.
(Option #2): Regular bloodwork for serum potassium and kidney function is critical, but ECG is not used to prevent hyperkalemia.
(Option #3): These drugs are critical for treating FT's heart failure and reduce her risk of mortality.

TAKEAWAY/KEY POINTS:

There is a long list of medications that can induce hyperkalemia and bloodwork should be done at regular intervals to monitor potassium levels.

REFERENCES:

[1] Hollander-Rodriguez JC, Calvert JF. Hyperkalemia. *Am Fam Physician*. 2006;73(2):283-290.

[2] Elliot MJ, Ronksley PE, Clase CM, Ahmed SJ, Hemmelgarn BR. *Can Med Assoc J*. 2010;182(15):1631-1635. doi:https://doi.org/10.1503/cmaj.100461.

The correct answer is: Acetaminophen is the only safe over-the-counter oral analgesic for FT

Question 8
ID: 46768
Correct
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Which of the following patients can be diagnosed with acute kidney injury (AKI)?

Select one:

- A 42-year-old male with a decrease in SCr by 20 umol/L within 24 hours ✖
- A 31-year-old female with an increase in SCr by 16 umol/L within 48 hours ✖
- A 74-year-old male with an increase in serum creatinine by 1.3 times baseline within 5 days ✖
- A 66-year-old female with an increase in serum creatinine by 2 times baseline within 7 days ✔

Rose Wang (ID:113212) this answer is correct. This patient meets the definition for acute kidney injury (AKI), which requires an increase in SCr by ≥1.5 times the baseline within 7 days.

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To understand the definition of acute kidney injury (AKI).

BACKGROUND:

Acute kidney injury (AKI) occurs when there is a rapid decrease in kidney function. AKI can be classified based on where the damage or impairment is occurring in the kidney:

- *Pre-renal AKI* occurs when there is hypoperfusion of the kidney (the most common cause of AKI)
- *Intrinsic AKI* occurs when there is damage or impairment within the kidney
- *Post-renal AKI* occurs when a severe blockage beyond the kidney (e.g. in the ureter) causes waste buildup in the kidney

Possible causes of AKI

Impairment Location	Possible causes of AKI
Pre-renal AKI	• Hypovolemia • Increased vascular resistance • Reduced cardiac function • Systemic vasodilation
Intrinsic AKI	• Bilateral renal artery stenosis • Infection • Immune system dysfunction (e.g. lupus, IgA glomerulonephritis) • Nephrotoxic drugs
Post-renal AKI	• Blood clots • Improperly placed catheter • Kidney stones (nephrolithiasis) • Urogenital cancers

AKI is defined as any of the following:

- Increase in serum creatinine (SCr) by ≥26.5 umol/L within 48 hours, or
- Increase in SCr by ≥1.5 times the baseline within 7 days, or
- Urine volume of <0.5 ml/kg/hour for 6 hours

RATIONALE:

Correct Answer:

(Option #4): This patient meets the definition for acute kidney injury (AKI), which requires an increase in SCr by ≥1.5 times the baseline within 7 days.

Incorrect Answers:

(Option #1): This patient does not meet the definition of acute kidney injury (AKI), which requires an increase in serum creatinine (SCr) by ≥26.5 umol/L within 48 hours.
(Option #2): This patient does not meet the definition of acute kidney injury (AKI), which requires an increase in serum creatinine (SCr) by ≥26.5 umol/L within 48 hours.
(Option #3): This patient does not meet the definition of acute kidney injury (AKI), which requires an increase in SCr by ≥1.5 times the baseline within 7 days.

TAKEAWAY/KEY POINTS:

AKI is defined as any of the following: increase in serum creatinine (SCr) by ≥26.5 umol/L within 48 hours, or increase in SCr by ≥1.5 times the baseline within 7 days, or urine volume of <0.5 ml/kg/hour for 6 hours.

REFERENCE:

[1] KDIGO clinical practice guidelines for acute kidney injury. Kidney International Supplements. 2012;2(1). doi:10.1038/kisup.2012.1.

The correct answer is: A 66-year-old female with an increase in serum creatinine by 2 times baseline within 7 days

Question 9

ID: 38886

Which of the following is **NOT** a role of the kidneys?

Correct

Flag question

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Select one:

- Release erythropoietin to stimulate red blood cell production ✖
- Release renin to control blood pressure and electrolyte levels ✖
- Secrete antidiuretic hormone to stimulate water retention ✔

Rose Wang (ID:113212) this answer is correct. Antidiuretic hormone is secreted by the posterior pituitary in response to plasma osmolarity.

- Play a major role in regulating acid-base balance ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To understand the functions of the kidneys.

BACKGROUND:

The kidneys filter about half a cup of blood per minute and are responsible for the following:

- *Optimizing acid-base balance*
  - The kidneys secrete hydrogen into the forming urine in order to raise blood pH
- *Regulating electrolyte levels*
  - The kidneys' absorption and secretion of sodium is affected by the renin-angiotensin-aldosterone system (RAAS) and antidiuretic hormone (ADH)
  - Potassium moves in the opposite direction of sodium (e.g., potassium is excreted into the urine if sodium is reabsorbed)
  - Chloride is regulated along with sodium via sodium-chloride symporter (i.e., co-transporter) channels in the kidneys
- *Regulating calcium and phosphate levels*
  - If serum calcium levels are low, parathyroid hormone (PTH) is released and stimulates the kidney to reabsorb calcium into the blood and excrete phosphate into the urine
  - If serum calcium levels are normal or high, PTH level is reduced, resulting in calcium excretion into urine and reabsorption of phosphate
- *Vitamin D activation*
  - PTH stimulates renal conversion of 25-hydroxycholecalciferol (i.e., calcidiol) into 1,25-hydroxycholecalciferol (i.e., calcitriol)
  - Calcitriol is the active form of vitamin D, which increases gut uptake of calcium
- *Optimizing red blood cell production*
  - The kidneys release erythropoietin (EPO) in response to low oxygen levels in the filtered blood
  - EPO stimulates the bone marrow to produce more red blood cells
- *Regulating blood pressure*
  - Cells surrounding the kidney's afferent arteriole release renin in response to low blood pressure
  - Renin is converted to angiotensin I, which in turn is converted into angiotensin II
  - Angiotensin II is responsible for the immediate systemic vasoconstriction, as well as a sustained release of aldosterone
  - Aldosterone causes sodium reabsorption in the kidney, resulting in fluid retention and blood pressure increase
- *Getting rid of waste such as drug metabolites*

RATIONALE:

Correct Answer:

(Option #3): Antidiuretic hormone is secreted by the posterior pituitary in response to plasma osmolarity.

Incorrect Answers:

(Option #1): Erythropoietin is released from the kidneys and acts on bone marrow to stimulate red blood cell production.
(Option #2): Renin is secreted from the juxtaglomerular region of the nephron.
(Option #4): Kidneys regulate hydrogen and bicarbonate balance in the circulatory system.

TAKEAWAY/KEY POINTS:

The kidneys are responsible for regulating serum electrolyte levels, acid-base hemostasis, calcium levels, blood pressure, and getting rid of waste.

REFERENCE:

[1] OpenStax. *Anatomy & Physiology*. OpenStax CNX. 2016. http://cnx.org/contents/14fb4ad7-39a1-4eee-ab6e-3ef2482e3e22@8.24.

The correct answer is: Secrete antidiuretic hormone to stimulate water retention

Question 10

ID: 38887

Correct

Flag question

Send Feedback

FC is a 75-year-old male who has been admitted to the hospital with acute kidney injury (AKI). He lives at home alone and has a daughter who visits him occasionally. His serum creatinine (SCr) was 220 umol/L upon arrival to the hospital and he has not had any urine output in the past 24 hours. His daughter says that her father suffers from osteoarthritis and has been using over-the-counter ibuprofen excessively over the past few days. In addition to osteoarthritis, FC's past medical history is significant for hypothyroidism and hypertension. His current medications include levothyroxine 112 mcg once daily and ramipril 2.5 mg once daily, but the ramipril has been held since his arrival to hospital. He has no known allergies to medications. His height is 168 cm and his weight is 80 kg.

All of the following options are appropriate monitoring parameters for FC during his hospitalization **EXCEPT**:

Select one:

- Urine output ✖
- Thyroid stimulating hormone (TSH) level ✔

Rose Wang (ID:113212) this answer is correct. Thyroid stimulating hormone (TSH) is an acute phase reactant and may be elevated from an acute kidney injury (AKI); however, monitoring is not necessary as this is only transient and may lead to inappropriate therapy modification.

- Serum potassium ✖
- Blood pressure ✖

Correct

Marks for this submission: 1.00/1.00.

TOPIC: Acute kidney injury (AKI)

LEARNING OBJECTIVE:

To identify the monitoring parameters for acute kidney injury (AKI).

BACKGROUND:

Once the offending agent has been removed from someone with acute kidney injury (AKI), kidney function should begin to return to normal levels and this will be reflected in decreased levels of serum creatinine (SCr). However, note that changes in SCr may lag behind changes in kidney function by 1-2 days. During management of acute renal failure, it is important to continually monitor serum potassium, hydration status, and blood pressure (target mean arterial pressure or MAP ≥65 mmHg). Continue to monitor the patient's fluid intake and urine output to assess for dehydration and fluid overload. Continue to monitor blood glucose levels especially in critically ill patients with a target blood glucose of 6-10 mmol/L.

Patients who have unstable kidney function, are unable to properly take in fluids, and/or are dehydrated, may be advised to undergo sick day management principles. Sick day management involves stopping certain medications (SADMANS) because these medications rely on adequate kidney function and continuing them could increase a patient's risk of adverse events. The following medications include sulfonylureas, ACE inhibitors, diuretics, metformin, ARBs, NSAIDs, and sodium glucose co-transporter inhibitors. In addition, patients who are on insulin require regular blood glucose monitoring and need to adjust their insulin accordingly.

RATIONALE:

Correct Answer:

(Option #2): Thyroid stimulating hormone (TSH) is an acute phase reactant and may be elevated from an acute kidney injury (AKI); however, monitoring is not necessary as this is only transient and may lead to inappropriate therapy modification.

Incorrect Answers:

(Option #1): Urine output should be monitored and expected to increase daily as kidney function returns to normal.
(Option #3): Potassium levels should be monitored as there may be an imbalance due to the acute kidney injury (AKI).
(Option #4): Blood pressure should be monitored, especially considering that the antihypertensive has been held.

TAKEAWAY/KEY POINTS:

Patients with acute kidney injury (AKI) should be monitored for serum creatinine (SCr), serum potassium, blood pressure, fluid intake, urine output, and blood glucose among other parameters during their hospitalization.

REFERENCE:

[1] Flurie RW. Disorders of Potassium and Magnesium Homeostasis. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e.* McGraw-Hill.
[2] Kantorvich, A. Medications That Always Use Actual Body Weight to Calculate Creatinine Clearance. *PharmacyTimes*. Published June 3 2016.
[3] Kidney Disease: Improving Global Outcomes (KDIGO) Acute Kidney Injury Workgroup. KDIGO clinical practice guideline for acute kidney injury. *Kidney Int Suppl.* 2012;2:1–138.
[4] Maker J, Roller L, Dager W. Acute Kidney Injury. In: DiPiro JT, Yee GC, Posey L, Haines ST, Nolin TD, Ellingrod V. eds. *Pharmacotherapy: A Pathophysiologic Approach, 11e.* McGraw-Hill.

The correct answer is: Thyroid stimulating hormone (TSH) level

Finish review